

Amendments to the Specification:

Please insert the attached paper copy of the Sequence Listing into the Specification.

Please replace paragraph 3, beginning on page 1, line, 27, with the following amended paragraph:

The observed inhibition of HSL by nicotinic acid treatment is mediated by a decrease in cellular cyclic adenosine monophosphate (cAMP) caused by the G-protein-mediated inhibition of adenylyl cyclase. Recently, the G-protein coupled receptors HM74 and HM74A have been identified as receptors for nicotinic acid (PCT patent application WO02/84298; Wise et. al. J Biol Chem. 2003 **278** (11) 9869-9874). The DNA sequence of human HM74A may be found in Genbank; accession number AY148884 (SEQ ID NOS: 1 and 2). Two other papers support this discovery, (Tunaru et. al. Nature Medicine 2003 (3) 352-255 and Soga et. al. Biochem Biophys Res Commun. 2003 **303** (1) 364-369), however the nomenclature differs slightly. In the Tunaru paper what they term human HM74 is in fact HM74A and in the Soga paper HM74b is identical to HM74A. Cells transfected to express HM74A and/or HM74 gain the ability to elicit G_i G-protein mediated responses following exposure to nicotinic acid. In mice lacking the homologue of HM74A (m-PUMA-G) nicotinic acid fails to reduce plasma NEFA levels.

Please replace paragraph 3, beginning on page 10, line, 12, with the following amended paragraph:

In-vitro testing

For transient transfections, HEK293T cells (HEK293 cells stably expressing the SV40 large T-antigen) were maintained in DMEM containing 10% foetal calf serum and 2mM glutamine. Cells were seeded in 90mm culture dishes and grown to 60-80% confluence (18-24h) prior to transfection. Human HM74A (GenBank™ accession number AY148884) (SEQ ID NOS: 1 and 2) was subcloned in to a mammalian

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expression vector (pcDNA3; Invitrogen) and transfected using Lipofectamine reagent. For transfection, 9µg of DNA was mixed with 30µl Lipofectamine in 0.6ml of Opti-MEM (Life Technologies Inc.) and was incubated at room temperature for 30min prior to the addition of 1.6ml of Opti-MEM. Cells were exposed to the Lipofectamine/DNA mixture for 5h and 6ml of 20% (v/v) foetal calf serum in DMEM was then added. Cells were harvested 48h after transfection. Pertussis toxin treatment was carried out by supplementation into media at 50ngml⁻¹ for 16h. All transient transfection studies involved co-transfection of receptor together with the G_{i/o} G protein, G_{o1}α.